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ON THE DETERMINATION OF INCOHERENT SCATTERING FUNCTION S (V)

THE deviation of the differential cross-sections for the incoherent scattering of gamma-rays from the Klein-Nishina predictions become significant for low energy photons and small scattering angles where the momentum transferred (q) to the electron is comparable with the momentum of the electron's motion within the atom. We have measured the values of incoherent scattering function S(V), which describes the effect of binding and motion of the scattering electrons for 145 keV gamma-rays and report the result in this letter.

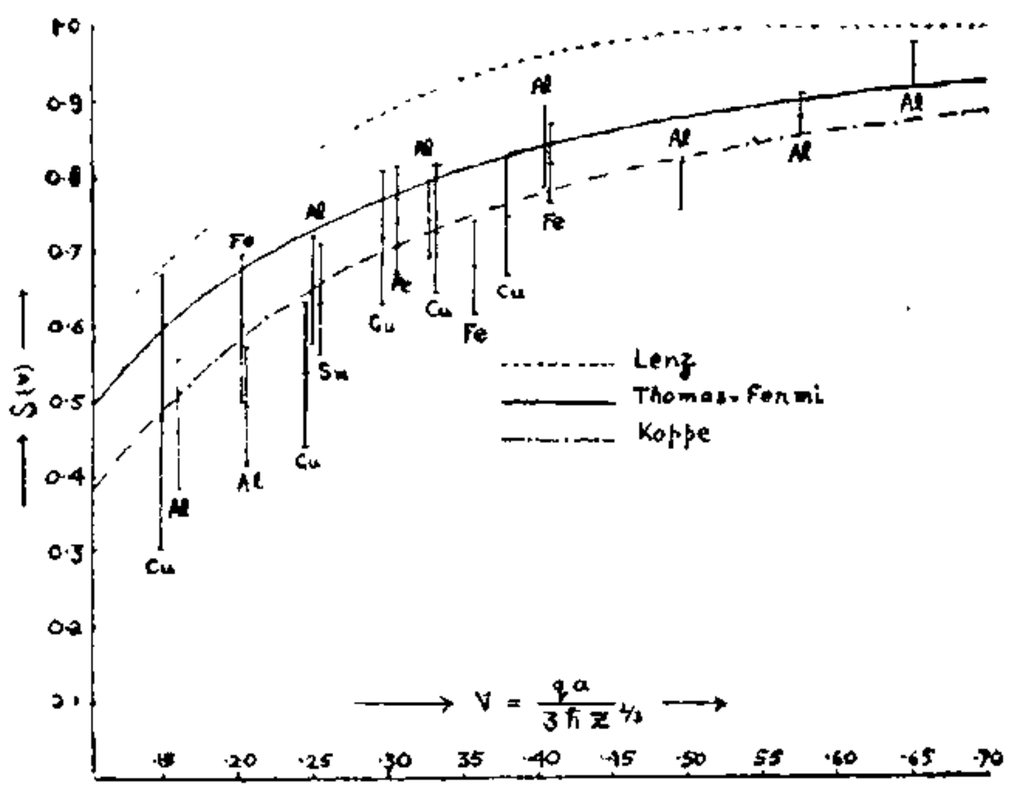

FIG. 1. Incoherent scattering function S (V) as a function of V.

The experimental arrangement is similar to our earlier measurements.[1] Gamma-rays of 145 keV energy from Ce^{141} were scattered from targets of tin, iron, copper and aluminium in the form of circular annular rings. The scattering angle was varied from 3° to 16° by changing the source detector distance while keeping the scatterer in the centre. As the energy of the incoherent scattered gamma-rays is almost the same as that of the coherent scattered gamma-rays, the result obtained gave total cross-sections (incoherent + coherent). From the total cross-sections the accurately known calculated contributions of coherent scattering[2] was subtracted. The ratio of the experimentally determined incoherent scattering cross-sections to the Klein-Nishina cross-sections gave the value of S(V). The results obtained are shown in Fig. 1 along with the theoretical[3-7] curves calculated for different electron distributions. The large errors at small value of V are due to the relatively large contribution of elastic scattering at small scattering angles.

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CHEMICAL COMPONENTS OF *DALBERGIA LANCEOLARIA* (FLOWERS AND LEAVES)

Dalbergia lanceolaria commonly known as Gaurakha is cultivated in the hilly areas of Mirzapur and Varanasi districts of U.P. in India. The leaves of this tree are used as a drug in the Indian medicine for the treatment of arthritis. Recent studies,[1] using animals and humans at Banaras, confirm that these leaves have beneficial effects in certain types of rheumatism and also possess anti-inflammatory properties. It has been reported that patients with serious lesions, where function of joints were completely impaired, have improved. We have therefore carried out a detailed chemical investigation of the leaves and flowers of *D. lanceolaria*.

Flowers.—Air-dried flowers (1 kg.) were successively extracted with light petroleum, acetone and alcohol in the soxhlet (64 hr. for each solvent). The light petroleum extractives were found to consist of waxy material. The green acetone and alcoholic extracts were found to contain the same components (T.L.C.); they were combined and concentrated to 300 ml. (approx). The concentrate when kept in the refrigerator for two days deposited a crystalline

solid (20 g.). This was boiled with methanol, filtered, and recrystallised from glacial acetic acid yielding a crystalline colourless solid, T.L.C. pure, m.p. 297–98°. It did not give any colour with alcoholic ferric chloride or Mg and HCl but when it was treated with sodium amalgam in ethanol, left overnight and acidified, a deep pink colour developed showing its isoflavone nature. It gave a deep blue colour when heated with gallic acid and concentrated sulphuric acid at 80° showing the presence of methylene-dioxy group ; u.v. spectrum $\lambda_{max.}^{MeOH}$ 256 mμ (log ε 4·67), 293 mμ (log ε 4·06). The above physical as well as chemical study revealed that the compound is ψ-baptigenin (7-hydroxy 3', 4' methylene-dioxy isoflavone). The identity was confirmed by the preparation of its methyl ether and acetate.

Leaves.—Air-dried leaves (800 g.) were similarly extracted with the above-mentioned series of solvents. The light petroleum extract gave only wax and chlorophyll. The acetone and alcoholic extracts gave ψ-baptigenin in quite high yields (2%). After removing it, the mother liquors were treated with neutral and basic lead acetates ; lead salts yielded quercetin and kæmpferol in low yields.

Since ψ-baptigenin was present in considerable amounts in the leaves of *D. lanceolaria* it was considered desirable to test whether it could be the main compound responsible for the beneficial properties of the leaves in arthritic ailments. A large sample was submitted to the Post-Graduate Institute of Indian Medicine and the report of tests[2] on animals and clinical trials on human patients confirm its potency.

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EFFECT OF ADRENERGIC BETA RECEPTOR BLOCKING DRUGS ON RAT BRAIN 5-HT LEVEL

Adrenergic β-receptor blocking drugs possess important effects on the central nervous system. Propranolol has been reported to possess central depressant and muscle relaxant as well as antitremor actions.[6,7,2] N-isopropyl-β (4-methanesulphonamidophenyl) ethanolamine (MJ 1999) also possesses a depressant action on the brain.[5] A recent adrenergic β-receptor blocking agent d-n-isopropyl-p-nitrophenyl ethanolamine (d-INPEA), however, has central excitatory effect.[6] Propranolol[5] and d-INPEA (unpublished observation) inhibit monoamine oxidase (MAO) activity, *in vitro*. Since this enzyme is concerned with the destruction of 5-hydroxy-tryptamine (5-HT), an amine which might be concerned with the functional activity of the brain, the present work was undertaken to study the effect of adrenergic β-receptor blocking drugs on brain 5-HT level in albino rats.

Propranolol, MJ 1999, d-INPEA or normal saline were injected intraperitoneally into albino rats (150–200 gm.). The animals were decapitated 1 hour later, after the behavioural effects of the drugs manifested, and their brains put in ice-cold acetone. 5-HT was extracted by the method of Amin, Crawford and Gaddum (1954)[3] and assayed on the rat stomach fundus by the method of Vane (1957).[8]

The effects of drug treatment on brain 5-HT level has been shown in Table I.

Table I
Effect of adrenergic β-receptor blocking drugs on rat brain 5-HT level

Drugs	Dose mg./kg.	No. of experiments	Brain 5-HT content μg./gm.	p value
Control ..	..	10	0·40	..
Propranolol	10	6	1·05	<0·05
MJ 1999 ..	80	6	0·92	<0·1
d-INPEA ..	80	6	0·45	>0·9

From Table I it may be seen that propranolol and MJ 1999 cause a rise in brain 5-HT. These drugs have also been reported to depress the central nervous system. However, only propranolol has been shown to possess MAO inhibiting action. It therefore appears that the rise in brain 5-HT may be due to the non-specific depressant action. Such an effect has also been reported after other sedatives like barbiturates, meprobamate and morphine.[1] The greater increase in 5-HT after propranolol may be due to its additional enzyme inhibiting effect. d-INPEA, in spite of its inhibiting action on MAO *in vitro*, does not elevate brain 5-HT which could be due to lack of enzyme inhibition, *in vivo*.

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